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OXYGEN DURING REST, HEAVY WORK, AND
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THE EFFECTS OF BREATHING 100 PER CENT OXYGEN
DURING REST, HEAVY WORK, AND RECOVERY

A Dissertation

Presented in Partial Fulfillment of the Requirements for
the Degree Doctor of Philosophy in the Graduate
School of The Ohio State University

by

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The Ohio State University
1964

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To Marjorie

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TABLE OF CONTENTS

CHAPTER	PAGE
I. INTRODUCTION	1
Statement of the Problem	9
Hypothesis	9
Limitations of the Study	9
Definition of Terms	9
Significance of the Study	10
II. SURVEY OF RELATED LITERATURE	11
Summation of Related Literature	43
III. METHODS AND PROCEDURES	49
Apparatus	50
Procedures	51
IV. ANALYSIS OF DATA	55
Description of Data	55
V. SUMMARY AND CONCLUSIONS	69
Summary	69
Conclusions	71
Recommendations	72
APPENDIXES	73
BIBLIOGRAPHY	87

LIST OF TABLES

TABLE	PAGE
1. Summary of the More Pertinent Studies: Effects of Breathing Pure Oxygen During Rest, Exercise, and Recovery	47
2. Age, Height, and Weight of Subjects	50
3. Gross Oxygen Cost for Each of Two Conditions in ML.	56
4. Mean Heart Rates (Beats/Min.) Per Time Period and Standard Deviations	57
5. Analysis of Variance-Gross Oxygen Cost	59
6. Mean Differences of Heart Rates for Six Different Time Intervals for Each of Two Conditions Dur- ing Exercise and Recovery in Beats/Min.	67

LIST OF FIGURES

FIGURE	PAGE
1. Mean Heart Rates Plotted for 5 Minutes of Rest, 5 Minutes of Exercise, and 30 Minutes of Recovery	58
2. Percentage Increases and Decreases in Resting VO ₂ , Gross Oxygen Cost, Net Oxygen Cost, Resting Minute Ventilation Volume, and Exercise and Recovery Ventilation Volumes . . .	60

CHAPTER I

INTRODUCTION

Since it was first learned by Lavoisier in 1777 that life depended on the oxygen in our atmosphere, inquiring minds have attempted investigations in order to determine whether this gas possesses stimulative or restorative powers. Researchers interested in the physiology of muscular activity are no exception. The physiological benefits of breathing oxygen-enriched gas mixtures before, during, after, and between bouts of severe exercise have long been a topic of controversy among exercise physiologists, physical educators, and coaches. Scientific data concerning the stimulatory effects of high concentrations of oxygen inhaled during heavy work are neither plentiful nor consistent. The contradictory results of these data have provided a quandry for coaches and athletes attempting to ascertain the merit of the inhalation of pure oxygen.

A. V. Hill in 1923, experimenting with the oxygen requirement of running at different speeds, compared with oxygen intake, reported that in running, the oxygen requirement increases proportionally as the speed increases. The highest values of oxygen intake were reached as a result of running at maximum speed and once the oxygen requirement

becomes larger than the oxygen intake, the runner can no longer maintain this speed and fatigue rapidly ensues.¹ The ability of man to take in and utilize oxygen is normally affected by the condition of the respiratory and cardiovascular systems. There seems to be some disagreement among physiologists as to which of these systems exert the greatest influence on oxygen consumption. According to A. V. Hill, the amount of oxygen passing into the blood and carried to and utilized by the working muscles depends on: (1) the degree of saturation of arterial blood, (2) the degree of saturation of the mixed venous blood, (3) oxygen capacity of the blood, and (4) the circulation rate.² Åstrand suggests that oxygen transport during muscular work is limited by pulmonary ventilation, rate of diffusion of oxygen from the alveoli to blood, minute volume of the heart, rate of blood flow through the working muscles, and the amount of oxygen diffusion from capillaries to the tissue.³

¹A. V. Hill, Muscular Activity (Baltimore: The Williams and Wilkins Company, 1926), p. 98.

²A. V. Hill, C. H. N. Long, and H. Lupton, "Muscular Exercise, Lactic Acid, and the Supply and Utilization of Oxygen," Royal Society of London Proceedings, (Series B, 97: Parts VII-VIII, 155-176, 1924), p. 163.

³P. O. Åstrand, "Human Physical Fitness With Special Reference to Sex and Age," Physiological Reviews, 36: 307-335 (July, 1956), pp. 316-317.

Bartlett and Hertz have stated that only under abnormal circumstances, such as the impedance of oxygen transfer across the alveolar membrane, are the effects of oxygen administration significantly obvious. It is believed that the physical association of oxygen with hemoglobin is such that hemoglobin is completely saturated with oxygen at the oxygen tension which exists in our environment and increasing the oxygen does not increase the oxygen content of the blood to an important extent.⁴ Wood, investigating normal oxygen saturation of arterial blood during the inhalation of either air or oxygen, found that breathing oxygen and air at sea level produced mean arterial oxygen saturations of 99.1 and 97.9 per cent respectively. It was considered that an oxygen tension of 150 mm of Hg. is sufficient to produce complete saturation of blood hemoglobin, and that an increase of oxygen tension to 600 mm Hg., as a result of breathing pure oxygen, should cause 100 per cent saturation of arterial blood. However, because of the low reported values following oxygen breathing, it was assumed that a systematic error was made in either the analytical procedures or in the calculation of physically dissolved oxygen.⁵

⁴Roscoe G. Bartlett and Rita A. Hertz, "Is It Possible to Differentiate by Inhalation Between Air and Oxygen?," Aerospace Medicine, 33: 552-556 (1962), p. 552.

⁵Earl H. Wood, "Normal Oxygen Saturation of Arterial Blood During Inhalation of Air and Oxygen," Journal of Applied Physiology, 1: 567-574 (1949), p. 568.

Studies concerned with investigating the effect of exercise on arterial oxygen saturation are limited. Hill, Long, and Lupton reported that during heavy muscular work the arterial blood leaves the lungs incompletely saturated, but not to the extent which might cause oxygen deficiency at the tissue level.⁶ Recent unpublished data collected in the exercise physiology laboratory at The Ohio State University concerning the physiological responses of conditioned athletes to heavy work loads indicate that there is no appreciable difference in the arterial oxygen tension at rest or during strenuous work. It has already been reported that unless the arterial oxygen saturation falls below 85 per cent, the oxygen demands at the tissue level are not seriously affected.⁷ Lillenthal, Riley, Proemmel, and Franke carried out direct measurements of arterial oxygen tension on one subject during heavy exercise at sea level and found that arterial oxygen tension fell from 94 mm of Hg. at rest to 73 mm of Hg. during exercise on three separate occasions.⁸ Filley also reports a PO_2 gradient between

⁶Hill, Long, and Lupton, op. cit., p. 161.

⁷John W. Bean, "Effect of Oxygen at Increased Pressure," Physiological Reviews, 25: 2-147, 1945, pp. 44-46.

⁸J. S. Lillenthal, R. L. Riley, D. D. Proemmel, and R. E. Franke, "An Experimental Analysis in Man of the Oxygen Pressure Gradient From Alveolar Air to Arterial Blood During Rest and Exercise At Sea Level and At Altitude," American Journal of Physiology, 147: 199-216, 1946, p. 201.

alveolar air and peripheral arterial blood of 9 - 10 mm of Hg. at rest and 16 - 20 mm of Hg. during moderately heavy work.⁹

Oxygen and carbon dioxide tensions of arterial blood were determined in fourteen young male athletes, at rest and during muscular exercise of varying intensities by Holmgren and Linderholm. The mean value for resting PaO_2 was reported as 100.9 mm of Hg., while the mean PaO_2 decreased significantly during heavy work to 84.9 mm of Hg. During exhaustive work the mean PaO_2 decreased further to a value of 78.8 mm of Hg. The difference between the value obtained during heavy work and that during exhaustive work was statistically significant.¹⁰

Having evaluated the research dealing with hypoxia during exhaustive work, the question arises as to whether inhalation of pure oxygen will aid in prolonging the ability to perform strenuous exercise and if so, can this work be completed at a lower energy cost than when breathing air from the atmosphere.

⁹Giles F. Filley, Gregoire Fernand, and George W. Wright, "Alveolar and Arterial Oxygen Tension and the Significance of the Alveolar-Arterial Oxygen Tension Difference In Normal Men," Journal of Clinical Investigation, 33: 517-529, 1954, p. 527.

¹⁰A. Holmgren and H. Linderholm, "Oxygen and Carbon Dioxide Tensions of Arterial Blood During Heavy and Exhaustive Exercise," Acta Physiologica Scandinavica, 43: 203-215, 1958, p. 206.

It is a well known fact that for a number of years coaches have recommended inhalation of pure oxygen to improve an individual's or team's performance or hasten their recovery. The Japanese swimming victories during the 1936 Olympics were attributed, at least in part, to breathing oxygen and aroused a great deal of interest in the effects of oxygen inhalation upon athletic performance.¹¹

In the 1960 Winter Olympic Games, the United States ice hockey team was trailing Czechoslovakia 4 - 3 after two periods in the final and decisive game for the team championship. During the first two periods the American team seemed slow moving and appeared listless. Upon recommendation of the Russian hockey captain, pure oxygen was administered to the American players between the second and third periods. Following this, the team's performance improved to the extent that they were able to score a resounding victory over their opponents.¹² It is common knowledge that both amateur and professional athletic teams administer 100 per cent oxygen to their players during brief periods of recovery. It is also well known that for a number of years oxygen self-contained breathing apparatus have

¹¹Peter V. Karpovich, Physiology of Muscular Activity (Baltimore: The Williams and Wilkins Company, 1926), p. 323.

¹²William Leggett, "Our Never-Say-Die Hockeymen," Sports Illustrated, 12: 22 (March 7, 1960), p. 22.

been used safely and successfully in fire fighting. Battle weary allied pilots in World War II were presumed "refreshed" by oxygen breathing before embarking on further assignments.

Karpovich studied the effects of oxygen inhalation on varsity swimmers before exercise and during recovery. Preliminary breathing of pure oxygen followed immediately by swimming a 100 yard sprint resulted in definite improvement of performance times over those times recorded for the same distance after breathing normal air. However, it was explained that the stimulating effects of oxygen on swimming speed was a result of being able to perform fewer respiratory movements during the actual swim, thereby increasing speed. It was also reported that the effect of preliminary oxygen breathing was more effective before swims of 100- to 220- yards than for longer distances. This was deemed probable because the swimmer was able to tolerate a larger oxygen debt and in doing so, reduced fatigue and increased the quality of his performance. Oxygen administered four to five minutes before swimming a 100-yard race proved to have no beneficial effect upon performance times. Although the subjects seemed to receive more rapid relief from symptoms of respiratory circulatory distress, breathing of oxygen for five minutes after a 100-yard swim appeared to have little or no effect on recovery. The ability to recover was based on swimming a second 100-yard sprint twenty minutes later and then comparing the performance times of

each swim. Karpovich, therefore, concludes that preliminary oxygen breathing immediately before a short race, during limited bouts of strenuous exertion, and during the first few minutes of recovery, is beneficial.¹³

Although man has the ability to store some oxygen at rest, and this store is increased as alveolar oxygen concentration is raised, this storage ability is considerably reduced during exercise. In the 1936 Olympics the Japanese in an attempt to increase their oxygen stores, terminated oxygen breathing more than five minutes before they entered the water. However, because of the five minute time lapse it can be assumed that oxygen was in no way responsible for their resounding victories in aquatics. Bartlett and Hertz recommend the use of oxygen before certain specific short contests when the ability to accumulate an oxygen debt is an important factor in determining the winner. However, any benefit derived from oxygen breathing before contests of longer duration is suspected to be psychological in nature.¹⁴

¹³Peter V. Karpovich, "The Effect of Oxygen Inhalation on Swimming Performance," The Research Quarterly, 5: 24-30, 1934, pp. 27-29.

¹⁴Bartlett and Hertz, op. cit., p. 552.

Statement of the Problem

The purposes of this study are threefold: (1) to ascertain the effects of breathing 100 per cent oxygen at rest, (2) to determine energy expenditure of conditioned athletes performing a heavy work load while breathing 100 per cent oxygen, and (3) to observe the effects of breathing pure oxygen on recovery.

Hypothesis

Based on previous studies concerning the effects of oxygen breathing before, during, and after strenuous exertion, it is felt that inhalation of oxygen will have no significant beneficial effect on energy expenditure while performing a heavy work load.

Limitations of the Study

1. Four experienced track men, one varsity soccer player, and one freshman swimmer were used as subjects.
2. Each subject was restricted to one trial during which he performed the required exercise and became acquainted with the experimental apparatus and procedures.
3. Each subject exercised once under each of the two conditions.

Definition of Terms

1. Pure oxygen, often referred to as Tank Oxygen or Medical Oxygen, is in essence 99.4 to 99.8 per cent oxygen mixed with nitrogen.

2. Respiratory distress as used in text refers to shortness of breath or a difficulty in breathing; dyspnea.

3. Pre-oxygenation period is defined as the fifteen minutes of pre-rest (oxygen experiments) used to wash out the alveoli.

Significance of the Study

Inquiries into the controversial topic of whether breathing pure oxygen sustains the athlete or relieves the fatigue of the industrial worker have yielded contradictory and inconclusive results.

It is hoped that further investigation pertaining to the effects of oxygen breathing during periods of severe exertion and its recovery might be of value to the designers and manufacturers of mine-rescue and firefighting equipment. This study might also be considered valuable in determining whether administration of increased concentrations of oxygen benefit the worker, soldier, or athlete in performing their physical tasks more economically.

CHAPTER II

SURVEY OF RELATED LITERATURE

In 1909, Douglas and Haldane compared the effect of forced breathing of normal air with quiet oxygen breathing in relation to their respective effects upon exercise. The pulse rate was slower and reached a normal value more quickly after breathing normal air before the exercise. The conclusions favored forced breathing of normal air as there was little or no evidence to support the theory that oxygen inhalation before heavy work benefits performance.¹⁵

Pembrey and Cook investigating the effects of oxygen inhalation upon man during rest and directly after exercise, raised the question whether factors other than carbon dioxide did not play a role in the development of dyspnea. At rest oxygen appeared to have little or no effect upon respiration but relieved symptoms of distress and postponed fatigue when breathed directly after exercise. The breathing of oxygen and subsequent postponement of fatigue led to the supposition that hyperpnea produced by muscular exercise is due in part to products of metabolism, other than carbon dioxide.¹⁶

¹⁵C. G. Douglas and J. S. Haldane, "The Causes of Periodic or Cheyne-Stokes Breathing," Journal of Physiology, 38: 401-419, 1909, p. 407.

¹⁶M. S. Pembrey and F. Cook, "The Influence of Oxygen Upon Respiration," Journal of Physiology, 37: 41-42, 1908, p. 37.

L. Hill and Flack made a number of post-exercise observations on pulse, blood pressure, and respiratory parameters on subjects who had breathed pure oxygen prior to the exercise. After breathing oxygen, each subject's athletic performance was improved and post-exercise data indicate a lowered pulse-frequency and a maintenance of a high blood pressure. It was suggested that fatigue following an athletic performance is primarily caused by cardiac deficiency due to the heart's lack of oxygen and that an increased concentration of oxygen in the inspired air improves the vigor of the heart.¹⁷ L. Hill and MacKenzie experimented with forced breathing of pure oxygen before a race of a half mile and a sprint of 250 yards. They discovered that not only did performance times improve, but during recovery the subject's pulse was stronger and he felt better and more refreshed than when he performed the same exercise after breathing air.¹⁸

As a result of his research on mine rescue apparatus, Briggs found that unconditioned subjects greatly benefited from breathing oxygenated air during physical exertion, but that their conditioned counterparts received

¹⁷Leonard Hill and Martin Flack, "The Influence of Oxygen on Athletes," Journal of Physiology, 38: 28-36, 1909, p. 30.

¹⁸Leonard Hill and J. MacKenzie, "The Effect of Oxygen Inhalation on Muscular Exertion," Journal of Physiology, 39: 33-35, 1909, p. 34.

no such advantage. An attempt was made to define physical fitness in terms of the results obtained from the comparison of exhaled carbon dioxide percentages when the subject breathed air and high oxygen mixtures. Curves representing carbon dioxide percentages of unconditioned subjects under both conditions exhibit a broad divergence while percentage curves for conditioned subjects were almost concurrent. It was concluded that fitness is inversely proportional to the degree of divergence of the two carbon dioxide curves. Data obtained from working at the most strenuous work loads demonstrated that even highly conditioned subjects derived some assistance as a result of oxygen breathing, while unconditioned subjects experienced an even greater advantage. However, regardless of the strenuousness of the exercise or the condition of the subject, oxygen enrichment of the inspired air above 60 per cent failed to yield any more of a significant effect than when breathing pure oxygen. Oxygen consumptions were calculated at rest and during work loads of varying intensities on a bicycle ergometer while breathing both normal air and oxygen-enriched gas mixtures. Oxygen intakes were found to increase significantly under the latter condition. Walking, running, and climbing tests were also carried out in order to determine the effect of breathing oxygenated air on efficiency of subjects performing these tasks. Based on calculated oxygen consumptions, inhalation of oxygen resulted in a more efficient execution of these

chores. It was mentioned that computing oxygen uptake from the respiratory quotient and carbon dioxide output under conditions of high oxygen pressures submit the results to a high probable error. This was explained in view of the fact that determination of the respiratory quotient depends on the equalization of nitrogen inhaled and exhaled. Measurements of R. Q. are evidently more accurate when nitrogen exceeds the oxygen volume as is the case when breathing atmospheric air. Because of the inaccuracy of calculating oxygen consumptions from just the respiratory quotients and carbon dioxide outputs when the oxygen concentration exceeds the inert gas concentration, another method of evaluation was applied. It involved separate quantification of inspired and expired minute volumes as well as separate determinations of corresponding oxygen proportions. The difference between the combined inspired and expired measurements yields the required result.¹⁹

A. V. Hill, Long, and Lupton²⁰ conducted a series of experiments concerning the supply and utilization of oxygen during and after muscular exercise. They were of the opinion that any beneficial physiological effects attributed to

¹⁹Henry Briggs, "Physical Exertion, Fitness, and Breathing," Journal of Physiology, 54: 292-318, 1920, p. 310.

²⁰A. V. Hill, C. N. H. Long, and H. Lupton, "Muscular Exercise, Lactic Acid, and the Supply and Utilization of Oxygen," Royal Society of London Proceedings, Series B, 97: Parts IV-VI, 84-138, 1924, pp. 84-138.

oxygen breathing during the recovery process were without foundation. The recovery process, following a bout of severe exercise, seemed unaffected by breathing 45 per cent oxygen, while increasing the oxygen concentration during exercise proved only slightly beneficial. Based on previous studies conducted by these authors there seems to be no specific difference between time courses while recovering after breathing normal air or high oxygen pressures. However, a higher oxygen uptake was attained during muscular exercise when breathing oxygen-enriched gas mixtures and it was suggested that the effects of this higher oxygen consumption leave a smaller oxygen debt. This was believed to be responsible for a more vigorous recovery, especially in the initial phases. Although breathing high concentrations of oxygen during exercise seemed to have some stimulating effects on the recovery process, there was no evidence to support the theory that oxygen administered during the recovery period itself, hastens the relief of fatigue. In contradiction to what A. V. Hill and his associates had expected, an increase in oxygen debt was produced by inspiring oxygen-rich gas mixtures. They had expected to find a more perfect saturation of arterial blood thus leaving a smaller oxygen debt and consequently shorten recovery.

Two subjects who previously had never exceeded an oxygen uptake of 3.9 liters per minute during exercise while breathing normal air attained oxygen consumptions of 5.5 and

5.1 liters per minute while breathing 52 per cent oxygen performing the same exercise. This increase was believed to be too large to be explained by simply insuring a more complete saturation of blood in its passage through the lungs. It was stated that a possible regulatory mechanism may exist in the heart muscle itself or in the central nervous system which responds to an increased oxygen pressure in arterial blood. It is then probable that the heart is able to regulate its output with respect to the oxygen concentration in arterial blood via coronary vessels or by some reflex in organs and tissues (possibly in muscles) which are negatively affected by a deficient oxygen supply. In this way circulation can be retarded as soon as a serious degree of unsaturation occurs and vice-versa when arterial oxygen concentration is higher than normal.

Measurements of ventilation during oxygen experiments produced varying and inconsistent results. In some instances, the breathing of 50 per cent oxygen seemed to significantly reduce ventilation, while on other occasions, there was an increase. A definite decrease in the respiratory quotient was noted during and immediately following exercise. This was believed to be due, at least in part, to the effect of increased oxygen pressure in arterial blood on the respiratory center. The inhibitory effect of increased oxygen concentrations on this center leads to elimination of

less carbon dioxide, thus accounting for the lowered respiratory quotient.

A. V. Hill and his associates feel that preceding investigators examining the effects of high oxygen pressures with respect to exercise failed to realize some of the serious errors when dealing with these increased concentrations. Their sensitivity concerning these errors was based on the fact that minor mistakes in experimental measurements may lead to large proportional errors in the results. For example, only a slight mistake in the quantification of the inspired and expired oxygen and carbon dioxide fractions may result in faulty inspired and expired ventilation volumes. This argument was illustrated numerically in the form of a sample problem and further accentuated by a table demonstrating how the error in oxygen intake is magnified in proportion to the increase of oxygen percentage in the inspired air. According to Hill, it is possible to avoid the error under discussion by direct measurements of inspired and expired volumes. However, the difficulty arises with respect to the accuracy in measuring both inspired and expired volumes. An error of one liter in the quantification of the expired volume will lead to an inaccuracy of 660 ml. in the calculated oxygen intake, while an error of one liter in the measurement of the inspired volume leads to an error of 700 ml. If the inspired volume is calculated from the measured expired volume, as is the usual procedure, then both volumes are subjugated to the same error. The errors,

when measuring the expired side only, tend to cancel out. However, if measurements are taken on both sides, separately, the errors may add together rather than cancel out, furnishing total errors of up to about 30 per cent of the true oxygen intake.

Another possible error may ensue if the investigator assumes that the volume of inert gas expired for any length of time is precisely equal to that inspired. This type of error is realized when an experiment demands the breathing of varying concentrations of oxygen and is of little importance when the subject breathes the same oxygen pressure for a long period of time before the observation begins. Hill stated that it is, therefore, important, when dealing with high and varying concentrations of oxygen, to breathe each concentration long enough to insure complete equilibration between the body and its new gaseous environment before acquiring any respiratory measurements. In conclusion, the observed oxygen and carbon dioxide errors become serious only when high oxygen pressures are employed and are further magnified when the equilibrium between the body and its new gaseous environment is not achieved. A. V. Hill and his collaborators thought that these errors exert little or no influence in high oxygen intakes of muscular exercise and recovery, but that they can seriously affect measurements of resting oxygen consumption. It is therefore, with precaution, that Hill and his colleagues recommend the use of the

Douglas bag technique when the inspired gas mixtures contain oxygen concentrations greater than 50 per cent.

In 1926, Hewlett, Barnett, and Lewis compared the effects of breathing pure oxygen and normal air during exercise upon pulmonary ventilation and lactic acid accumulation in blood and urine. During the oxygen experiments minute volumes of respiration were reduced and concentrations of expired carbon dioxide were increased. There were no definite changes recorded in the lactic acid content of the blood or urine.²¹

In 1931, Miyama reported that breathing oxygen before and after a 120 meter run caused the subjects to sprint the distance faster and to recover more quickly than if normal air was breathed before the run. It was surmised that inhalation of oxygen had a stimulating effect before running because the greater content of oxygen in the blood may act more promptly in reducing fatigue in the muscles. This increased supply of oxygen will, therefore, be more than adequate to prevent accumulation of excess lactic acid

²¹A. W. Hewlett, G. D. Barnett, and J. K. Lewis, "The Effect of Breathing Oxygen-Enriched Air During Exercise Upon Pulmonary Ventilation and Upon the Lactic Acid Content of Blood and Urine," Journal of Clinical Investigation, 3: 317-325 (December 20, 1926), pp. 319-323.

in the muscles and circulatory system, believed by many to be the direct cause of fatigue.²²

In 1934, Christensen, Krogh, and Lindhard reported results of their studies on heavy work. It is interesting to note that when exercise was so strenuous that it could not be maintained for two minutes, the administration of oxygen had no effect on performance. However, if the subject began exercising at a moderate work load (breathing an increased concentration of oxygen) and the intensity was gradually increased by stages, then he was able to sustain rates of work which had previously exhausted him in two minutes. It was concluded that the difference in reaction of subjects to the inhalation of pure oxygen under the condition of gradual increase in work intensity might be explained in terms of a longer time requirement for the adjustment of the respiratory and circulatory systems to the high oxygen pressures.²³

²²Akira Miyama, "Some Pharmacological Experiments On The Recovery of the Circulatory System From Fatigue After Bodily Exercise, II, The Effect of Oxygen Upon Bodily Exercise," Acta Scholae Medicinalis, Kioto, 14: 73-78, 1931, p. 75.

²³E. H. Christensen, A. Krogh, and J. Lindhard, "Investigations On Heavy Muscular Work," Quarterly Bulletin Health Organization, L.O.N., 3: 388-417, 1934, p. 416.

Barach, also in 1934, stated that breathing pure oxygen, while performing a step test on an eight inch bench at the rate of forty times per minute, increased efficiency of performance, significantly lowered respiratory and pulse rates, and reduced pulmonary ventilation.²⁴

Nielsen and Hansen, studying the effects of breathing oxygen during heavy work, found that pulmonary ventilation was lowered considerably, while oxygen breathing during moderate work exhibited no effect whatsoever. Due to the fact that oxygen inhalation ~~should~~ have practically no effect on the proprioceptive reflexes in heavy work, it was assumed that there were other factors involved in the regulation of breathing under these conditions. The oxygen supply to the working muscles was lowered significantly during vigorous exertion and it was surmised that certain substances are liberated in the muscles under this partially anaerobic condition. It was believed that this condition was responsible for the increased pulmonary ventilation and that breathing oxygen affords a more adequate supply of this gas, diminishing the production of anaerobic metabolites, thus lowering ventilation.²⁵ Asmussen and Nielsen support

²⁴A. L. Barach, "The Action of Oxygen In Counter-acting Alcoholic Intoxication," American Journal of Physiology, 125: 90-117, 1954, p. 614.

²⁵M. Nielsen and O. Hansen, "Maximale körperliche Arbeit bei Atmung O₂ - reicher Luft," Scandinav. Archiv. Für Physiol., 76: 37-59, 1937, p. 40.

the results presented in the study by Nielsen and Hansen. It was the purpose of their research to determine the effects of breathing 100 per cent oxygen on the regulation of respiration during exercise. Ventilations while breathing oxygen during light work intensities were the same as, or a little higher than when normal air was breathed during identical work. However, as the strenuousness of exercise was increased, the pulmonary ventilation decreased. It was demonstrated, during oxygen experiments, that at an oxygen consumption of 1.1 liters per minute, ventilation began to decrease and became lower as the oxygen uptake was increased. Alveolar carbon dioxide tension was found to decrease only slightly during rest and at small work loads during oxygen inhalation. It became higher than in the corresponding air experiments when an oxygen consumption of 1.1 liters was reached and increased steadily up to a value of 47 mm of Hg. (at an $\dot{V}O_2$ of 2.5 liters/minute). Results from riding a bicycle ergometer at a work load of 1,440 mkg./min. breathing air, 30 per cent, 60 per cent, and 100 per cent oxygen indicated that as the oxygen concentration of inspired air was increased, there was significant and gradual lowering of the blood lactate level. Blood lactates decreased, during the experiment when the highest oxygen concentration was breathed, to 40 per cent of the value of the air experiment.

Asmussen and Nielsen stated that the reduction of the PO_2 during heavy work, while breathing air, is too diminutive to stimulate the chemoreceptors. Instead they proposed that some unidentifiable substance, correlating to lactic acid, is produced in the working muscles during anaerobiosis which causes an increase in pulmonary ventilation and that the effect of oxygen breathing on ventilation is due to the reduction of this substance. The possibility of blood lactate changes to produce this effect was excluded because of their slow rate of change. It is a common fact of knowledge that a reduction of arterial oxygen tension stimulates ventilation through its action on the chemoreceptors of the aortic and carotid bodies. It was suggested that if arterial oxygen tension were lowered significantly, then the effect of oxygen breathing might be explained as a result of the diminished activity of these chemoreceptors. However, oxygen tension never decreased seriously during these experiments and it was, therefore, concluded that the effect of oxygen breathing, with respect to ventilation during severe exertion, was not due to elimination of any oxygen deficiency in arterial blood.²⁶

²⁶Erling Asmussen and Marius Nielsen, "The Effect of Auto-Transfusion of Work Blood on the Pulmonary Ventilation," Acta Physiologica Scandinavica, 20: 79-87, 1950, pp. 173-178.

The concentration of blood lactic acid was observed by Lundin and Ström on subjects who were working on a bicycle ergometer and breathing nitrogen-oxygen mixtures with a varying percentage of oxygen. Data acquired on two well-conditioned subjects indicated that a reduced inspiratory oxygen partial pressure produced an abnormal increase in blood lactic acid for a given amount of work. However, a sudden increase in arterial oxygen pressure lowered the lactate level, reduced minute ventilation, and in the case of one subject, significantly decreased his oxygen consumption for that work period.²⁷

Dripps and Comroe conducted a series of experiments designed primarily to determine the threshold of normal human respiratory and circulatory systems to anoxemia. In one phase of the experiments, the subjects were asked to breathe varying mixtures of inspired air through a mask equipped with a ~~demand~~ regulator and valve. Effects of breathing medical oxygen were observed on such variables as pulse rate, cardiac output, stroke volume, arterial oxygen saturation, and respiratory minute volume without regard to exercise. An immediate effect of 100 per cent oxygen was observed in the slight reduction of resting

²⁷G. Lundin and G. Ström, "The Concentration of Blood Lactic Acid In Man During Muscular Work in Relation to the Partial Pressure of Oxygen of the Inspired Air," Acta Physiologica Scandinavica, 13: 253-266, 1947, p. 257.

minute ventilation and pulse rate. Continuous breathing of this gas mixture (six to eight minutes) produced an increase in pulmonary ventilation and significantly reduced heart rate and cardiac output. It was suggested that the stimulating effect of high concentrations of oxygen on minute ventilation might be attributed to an increase in partial pressure of carbon dioxide at the tissue level. This supposedly occurs because of the decrease of available reduced hemoglobin for carbon dioxide transport, an increase in medullary partial pressure of carbon dioxide as a result of cerebral vasoconstriction, an improvement in arterial oxygen tension at the medullary center, or lastly, by a lower respiratory tract reflex initiated by the irritating effect of pure oxygen. The latter possibility, because it has more factual supporting evidence, was considered to be the primary cause of increased ventilation. These experiments seemed to indicate that oxygen sensitive chemoreceptors in humans are continually active. With respect to respiration, it was believed that these receptors have only minimal activity under normal conditions and that their activity increases significantly only when inspired oxygen concentrations become seriously low, such as in times of extreme stress (as in the case of strenuous exercise).²⁸

²⁸Robert D. Dripps and Julius H. Comroe, "The Effect of The Inhalation of High and Low Oxygen Concentrations on Respiration, Pulse Rate, Ballistocardiogram and Arterial Saturation (Oximeter) of Normal Individuals," American Journal of Physiology, 149: 277-291, 1947, pp. 279-281.

Asmussen, Döbeln, and Nielsen investigated the effects of varying concentrations of oxygen in the inspired air on the ability to perform two different work loads. The capacity for the subjects to perform work was determined in two ways: one involved maximum work performed at a given rate of 2,000 mkg. per minute, while the other required the subject to attempt to perform a given amount of work (9,860 mkg.) in the shortest possible time. Two of the subjects working at the constant rate to exhaustion, compiled higher total work loads and worked at this rate longer as a result of breathing 100 per cent oxygen. The amount of time necessary to perform 9,860 mkg. of work was decreased as a result of breathing pure oxygen and slightly increased while inhaling a gas mixture with a low oxygen concentration. Oxygen debts and oxygen consumptions were calculated for the trials involving inhalation of low or normal oxygen pressures, but the same measurements were not presented for those trials where 100 per cent oxygen was used. Maximum blood lactates were also determined, but exhibited little change regardless of the type of work load performed or oxygen mixture breathed.²⁹

²⁹Erling Asmussen, W. von Döbeln, and Marius Nielsen, "Blood Lactate and Oxygen Debt After Exhaustive Work At Different Oxygen Tensions," Acta Physiologica Scandinavica, 15: 57-62, 1948, pp. 58-59.

Based on results of studies conducted in 1946³⁰ and again in 1948,³¹ Asmussen and Nielsen concluded that there is a probability that reflexes from moving limbs assume a regulatory role in the control of respiration during exercise. They proposed that during heavy work, substances are produced within the working muscles which stimulate the respiratory center directly or indirectly through their action on the chemoreceptors and, therefore, cause an increase in ventilation. Experiments with oxygen breathing during heavy exercise indicated a diminution of ventilation, which was thought to be a result of the reduction of anaerobic substances by increased oxygen concentrations.

It was the purpose of this study to show, directly, that blood during severe exercise does contain such substances which exert a striking stimulatory effect on respiration. This was accomplished by auto-transfusion of the legs after cessation of heavy work with legs only. Immediately after exercise blood was trapped in the legs via the use of blood pressure cuffs and was not released until pulmonary ventilation attained a near-resting level. According to the authors, a significant increase in ventilation after the cuffs were deflated indicated a presence of substances in "work blood" which are thought to play a role

³⁰Asmussen and Nielsen, loc. cit., "Studies on the Regulation of . . ."

³¹Asmussen et. al., loc. cit., "Blood Lactate...."

in the control of respiration during exercise. "Work blood" was defined as that venous blood, containing anaerobic metabolites, which is trapped in muscles by auto-transfusion. When pure oxygen was breathed during the whole period of occlusion (15 minutes) and after the "work blood" was released, a more noticeable decrease in ventilation was observed than when breathing ordinary air.³² This finding was in agreement with results obtained in previous oxygen experiments conducted by Asmussen and Nielsen.

Asmussen, again in 1950, demonstrated conclusively that during prolonged exercise on a bicycle ergometer, the addition of oxygen to the inspired air produced a significant reduction in pulmonary ventilation, raised alveolar carbon dioxide tension, and decreased blood lactic acid concentration. Work loads of light or moderate intensities failed to yield similar results.³³

Hickam et al. reported respiratory and circulatory data on a number of healthy male medical students and physicians while they inspired gas mixtures containing different concentrations of oxygen. These data were collected

³²Erling Asmussen and Marius Nielsen, "The Effect of Auto-Transfusion of 'Work Blood' on the Pulmonary Ventilation," Acta Physiologica Scandinavica, 20: 79-87, 1950, pp. 82-86.

³³Erling Asmussen, "Blood Pyruvate and Ventilation in Heavy Work," Acta Physiologica Scandinavica, 20: 133-136, 1950, p. 135.

both at rest and during a vigorous run on a motor-driven treadmill. A segment of the experimental procedure consisted of switching the subject from room air to 15 per cent oxygen, back to air, and then finally to 100 per cent oxygen during exercise. Each gas mixture was breathed for one and one-half minutes, amounting to a total exercise time of six minutes. Each subject was completely naive with respect to the gas mixture they were breathing. Results of this study demonstrated a significant decrease in pulmonary ventilation, an effect reported previously by authors experimenting with the breathing of pure oxygen during exercise. Ventilation seemed to be affected quite rapidly by an increase in the inspired oxygen concentration, for it took only ten seconds or about four or five breaths of oxygen-enriched air to render its effect. There were also reports of an increase in carbon dioxide output and a rapid lowering of blood pH level. Rapid ventilatory response to an increased arterial oxygen saturation was thought by some researchers to be too immediate to be explained in terms of changes in the concentration of some substance in the blood which is produced by muscles during anaerobiosis. Instead, it was assumed that the human chemoreceptor system becomes highly sensitive to arterial oxygen tension or that a possibility exists in which the respiratory center reacts to a disturbance in the equilibrium or "tone" which, according to Hickam, is known

to prevail at normal oxygen tensions in the resting state. The latter theory was believed to better explain the increased oxygen tension's effect, as a variety of neurogenic stimuli become more active as the severity of exercise increases.³⁴ Gesell, in his discussion of the regulation of respiration by carbon dioxide, hydrogen ion concentration, and oxygen tension in arterial blood, stated that since the supply of oxygen determines the amount of lactic acid and carbon dioxide formed in living tissues and since it controls the transport and elimination of these waste products, oxygen tension in the arterial blood constitutes the indirect regulator of normal pulmonary ventilation.³⁵ Comroe and Schmidt contribute to this discussion by asserting that passive stretch reflexes play only a minor role in the regulation of normal respiration while the effect of neurogenic stimuli is vastly underestimated.³⁶

In 1952, Miller and his associates examined the effects of oxygen inhalation on the cardiovascular function of conditioned and unconditioned subjects during exercise

³⁴J. B. Hickam, W. W. Pryor, E. B. Page, and R. J. Atwell, "Respiratory Regulation During Exercise in Unconditioned Subjects," Journal of Clinical Investigation, 30: 503-516, 1951, pp. 510-513.

³⁵Robert Gesell, "On The Chemical Regulation of Respiration," American Journal of Physiology, 66: 5-49, p. 21.

³⁶Julius H. Comroe and C. F. Schmidt, "Reflexes From The Limbs As a Factor in Hyperpnea of Muscular Exercise," American Journal of Physiology, 138: 536-547, 1943, p. 546.

and recovery. Results of this study indicated that oxygen breathing had little or no influence on heart rate and systolic blood pressure during exertion. Maximum heart rates, recorded at the end of exhaustive work, exhibited only slight diversifications, regardless of the gas mixture breathed. Although administration of pure oxygen seemed to have no effect on unconditioned subjects in this experiment, a significant reduction of the concentration of lactic acid in the blood was noted in athletes during both moderate and exhaustive work. The ability of conditioned subjects to perform exhaustive work for longer periods of time was greatly improved during the oxygen experiments. However, the recovery process following this severe work was not altered by breathing increased concentrations of oxygen.³⁷

In 1954, Åstrand exposed a subject to high oxygen pressures and then carried out a number of respiratory measurements while the subject performed work of varying grades of severity. Similar experiments were also conducted utilizing normal atmospheric air. The Pulmonary ventilation measured during work, requiring an oxygen intake of not more than 1.4 liters per minute, was not modified by enrichment of the oxygen content of air. However, as the strenuousness of the exercise increased, requiring oxygen intakes as high

³⁷A. T. Miller, H. L. Perdue, E. L. Teague, and J. S. Ferebee, "Influence of Oxygen Administration on Cardiovascular Function During Exercise and Recovery," Journal of Applied Physiology, 5: 165-168 (Oct., 1952), p. 168.

as 1.5 to 4.0 liters per minute, the pulmonary ventilation attained was always less during the inhalation of oxygen than was the case when ordinary air was breathed. These results are in accordance with findings reported by Asmussen and Nielsen which were summarized earlier in this chapter.³⁸ Heart rates tended to be lower during oxygen experiments. These experiments were designed so as to ascertain the blocking effect of breathing pure oxygen on the chemoreceptors, the results of which seem to indicate that the hyperbaric "drive" from the chemoreceptors, as described by Åstrand, was definitely inhibited by higher concentrations of oxygen in the inspired air as was evident in the comparison of pulmonary ventilation values.³⁹

Varying the intensity of the exercise to coincide with their subjects' physical condition, Bannister and Cunningham compared the effects of breathing atmospheric air, 33, 66, and 100 per cent oxygen on respiration and physical performance. The intensity of exercise was chosen for each subject so that his breaking point was reached in six to nine minutes when breathing room air. Additional concentrations of oxygen in the inspired air above that of room air, improved physical performance considerably.

³⁸Asmussen and Nielsen, loc. cit., "Studies on the Regulation of . . ."

³⁹P. O. Åstrand, "The Respiratory Activity in Man Exposed to Prolonged Hypoxia," Acta Physiologica Scandinavica, 30: 343-368, 1954, pp. 348-349.

However, any increase in oxygen saturation above 66 per cent, failed to enhance performance times. Subjective results indicated that symptoms of respiratory distress and fatigue were alleviated more adequately by the inspiration of 66 per cent oxygen rather than the breathing of medical oxygen.⁴⁰ This result differs from others reported in the literature in that it demonstrates there is an optimum level for alveolar oxygen tension during heavy exercise and that if this level is exceeded, performance ability declines. It is suggested that the depressing effects of 100 per cent oxygen inhaled during vigorous exercise might be attributed to the increase in cerebral circulation resulting from the excess of circulating carbon dioxide and lactic acid. This increase prevents any protection from the injurious effects of high concentrations of oxygen afforded to the brain by cerebral vasoconstriction which occurs at rest when medical oxygen is breathed.

Determinations of pulmonary ventilations showed decreases between 5 to 25 liters per minute in all subjects when breathing pure oxygen. The blood lactate level was also reduced as oxygen concentration was increased. Measurements of alveolar partial pressure of carbon dioxide indicated a slight drop in this pressure as the exercise

⁴⁰R. G. Bannister and D. J. C. Cunningham, "The Effects on the Respiration and Performance During Exercise of Adding Oxygen to the Inspired Air," Journal of Physiology, 125: 90-117, 1954, pp. 118-131.

continued in contrast to its sharp rise when exercise first began. These data are based on results obtained during strenuous exercise. No significant changes in these variables were detected in light or moderate work. Oxygen uptakes were also reported, but were disclosed to be unreliable for reasons cited earlier in this chapter by A. V. Hill, Long, and Lupton.⁴¹

The data presented by Bannister and Cunningham are, at least partially, in agreement with those findings of A. V. Hill and his collaborators, who studied the effects of oxygen breathing in 1924.⁴² Hill added oxygen during heavy work and found, that although pulmonary ventilation did not always decrease, the capacity for maximum exertion was enhanced and there was a 20 per cent increase in oxygen consumption. Subjects who breathed pure oxygen during exercise were also able to tolerate higher concentrations of blood lactate. It was postulated that the differences are too great to be explained in terms of an increased oxygen content in arterial blood. This tolerance for higher lactate levels might be explained by the fact that during exhaustive exertion there is a reduction in oxygen tension in arterial blood which could affect the heart and brain. Bannister and Cunningham believed by adding oxygen, cardiac

⁴¹Hill, Long, and Lupton, op. cit., "Muscular . . .," Parts IV-VI, p. 89.

⁴²Ibid., p. 84.

output was increased, anoxemia was relieved, and an increased tolerance for acid metabolites was realized. It was also proposed that arterial anoxemia during exercise was caused by the depression of arterial oxygen tension, which in turn, is attributed to abnormal gaseous diffusion or distribution of gas or blood and the rapid utilization of oxygen by the working muscles during arduous work. It was conjectured that if the partial pressure of oxygen is depressed during heavy exercise, then the addition of a higher oxygen concentration in the inspired air should result in a rise in arterial oxygen pressure to values in excess of the threshold of the carotid and aortic chemoreceptors. This was believed to be the cause of the lowered pulmonary ventilation during exercise.⁴³

In another publication by Bannister et al., it was postulated that oxygen lack, lactic acid accumulation in muscles and blood, and carbon dioxide excess were the causative factors of hyperpnea in severe exercise. Although the effects of nervous stimuli from working muscles and joints cannot be disregarded, they are thought to contribute very little to the increase in ventilation. The

⁴³Bannister and Cunningham, loc. cit.

effectiveness of working limbs in respiratory control was believed to be realized at the beginning of exercise when rapid adjustments to changing conditions is necessary.⁴⁴

According to Åstrand, the partial pressure of carbon dioxide, hydrogen ion concentration, partial pressure of oxygen in arterial blood and venous blood, temperature of blood, nervous impulses (supposedly arising from receptors in working muscles and joints or the motor cortex), and metabolites e.g. blood lactic acid, are all to be considered as factors regulating respiration during work. However, it was believed that the increase of ventilation during work cannot be fully explained by these changes alone.⁴⁵ Asmussen and Nielsen suggested that nervous impulses and/or chemical substances, presumably anaerobic metabolites, assume the major role in controlling ventilation during exercise.⁴⁶

Åstrand stated that if nervous impulses from muscles or motor centers existed, then their activity should depend on the severity of the work because pulmonary ventilation, as previously demonstrated, increases rectilinearly with

⁴⁴R. G. Bannister, D. J. C. Cunningham, and C. G. Douglas, "The Carbon Dioxide Stimulus to Breathing in Severe Exercise," Journal of Physiology, 125: 90-117, 1954, p. 99.

⁴⁵Åstrand, op. cit., "Human Physical Fitness . . .", p. 319.

⁴⁶Asmussen and Nielsen, op. cit., "Studies on the Regulation . . .", p. 171.

the intensity of the work load. It was, therefore, difficult

. . . to explain the moderate difference in pulmonary ventilation that is seen during the first 10-15 seconds after the start of work, in one case of 300 kpm/min. and 2400 kpm/min. in the other, on a bicycle ergometer. In the latter case the nervous activity must be enormous compared to the experiment in which a work of 300 kpm/min. is performed. The final differentiation in pulmonary ventilation takes place after about 20 seconds, after which time the blood from the working muscles can be supposed to have reached the known chemoreceptors and the respiratory center. Furthermore, if a work load of 1200 kpm/min. is changed to work of a lower intensity, in one case 900 kpm/min., in the other 300 kpm/min., the decrease in ventilation registered during the first periods of five seconds is mainly similar . . .⁴⁷

These unpublished data suggested that nervous impulses play only a minor role in the regulation of respiration during muscular work. Instead, it was proposed that regulation was more likely to involve the circulatory system and stimulation of receptors via the blood.

Additional unpublished respiratory data assembled by Åstrand and his associates indicated that a decrease in ventilation occurred when air was replaced by 100 per cent oxygen during periods of severe exercise. It was suggested that hypoxia of peripheral arterial blood occurred during strenuous work and that adding a higher concentration of oxygen to the inspired air inhibited the hypoxic "drive" for increased pulmonary ventilation, which normally occurs

⁴⁷Åstrand, op. cit., "Human Physical Fitness . . .", pp. 319-320.

when ordinary air is breathed. The author stressed the fact that activity of the chemoreceptors depends on the oxygen tension, not the oxygen saturation of arterial blood.

Improvement of physical performances attributed to breathing pure oxygen have not been thoroughly investigated but it was mentioned in this publication that among other causes contributing to this improvement, the increase of the partial pressure of oxygen in arterial blood can be supposed to affect the heart muscle favorably, whereby its output increases.

Barrat-Boyes and Wood, using a group of healthy men, conducted a study in which the effects of breathing atmospheric air and 100 per cent oxygen on exercise in the supine position were compared. Oxygen administered at rest and during light work intensities resulted in only a slight decrease in heart rate and an insubstantial improvement of stroke volume. The cardiac output was also unaffected. An increase in systemic and a fall in pulmonary arterial pressure was noted under exercise conditions. On the basis of these data, it was concluded that the changes were too diminutive in degree and were considered insufficient to influence the human response to more strenuous exercise.⁴⁸

⁴⁸Brian G. Barrat-Boyes and Earl H. Wood, "Hemodynamic Response of Healthy Subjects to Exercise in the Supine Position While Breathing Oxygen," Journal of Applied Physiology, 11: 129-135, 1957, pp. 131-134.

The effects of inhalation of 100 per cent oxygen at one atmosphere were considered under five categories, without regard to exercise, by Baker and Hitchcock in 1957. Ventilation volume, carbon dioxide output, oxygen consumption, respiratory rate, and the resting respiratory quotient were determined on healthy male subjects during inhalation of pure oxygen and room air. The recent literature indicates an increase in ventilation volume, oxygen consumption, and carbon dioxide output while breathing medical oxygen at rest, but when the subject is returned to normal air, these parameters decline rapidly to their normal resting levels. These series of experiments were designed to ascertain if any respiratory compensation occurred when the subject was exposed to room air and pure oxygen. Increases in carbon dioxide output and ventilation volume during oxygen breathing were essentially the same with the rise in expired carbon dioxide percentage thought to be a result of the increased ventilation. It was postulated that the rapid rise in ventilation and respiratory rate was due to the stimulus of a higher carbon dioxide tension and a lower pH on the medullary respiratory center. It was suggested that the cause of increased carbon dioxide tension and subsequent reduction of blood pH under the condition of high oxygen pressure was due to the loss of part of the function of hemoglobin. An immediate effect on carbon dioxide output, oxygen consumption, and resultant respiratory quotient was

noted when the subjects were switched from pure oxygen to room air. Carbon dioxide output decreased 5.3 per cent, oxygen consumption was reduced 25.8 per cent, and the computed respiratory quotient exhibited an increase of 42.1 per cent. The explanation for this apparently lies in the fact that as more oxygen is physically dissolved in intracellular and extracellular body fluids, the greater the increase in the respiratory quotient. Oxygen consumptions were presumed increased as a result of breathing 100 per cent oxygen, but results of these determinations were not deemed precise enough to be reported.⁴⁹

The regulation of respiration during heavy work was studied in young normal subjects by Asmussen and Nielsen by measuring arterial oxygen pressure and various respiratory functions at rest and during work when breathing normal air and gas mixtures with augmented or lowered oxygen concentrations. As a result of their comparisons of oxygen tensions while breathing atmospheric air, 33 per cent oxygen, and 100 per cent oxygen, Asmussen and Nielsen concluded that hyperventilation in heavy work cannot be explained as a result of arterial hypoxia only. Instead, it was postulated that the diminished pulmonary ventilation during strenuous

⁴⁹Saul P. Baker and Fred A. Hitchcock, "Immediate Effect of Inhalation of 100 Per Cent Oxygen at One Atmosphere on Ventilation Volume, Carbon Dioxide Output, Oxygen Consumption and Respiratory Rate in Man," Journal of Applied Physiology, 10: 363-366, 1957, pp. 363-365.

exercise while breathing pure oxygen was due to the abolition of the chemoreceptive drive which, under normal conditions, increases the sensitivity of the respiratory center towards the work stimulus and partly to the anaerobic production of some substance which acts to increase ventilation. This active substance, although thought to correlate to lactic acid, was not believed to be lactic acid itself because the changes in ventilation were completed more quickly than the changes in blood lactate level both when switching from room air breathing to oxygen breathing and when switching back again from oxygen to air.⁵⁰

Brandis and associates conducted a series of experiments in which healthy mine workers exercised at a moderate work load breathing either room air or 96 per cent oxygen. The purpose of this investigation was to examine respiratory exchange and the state of the oxidation-reduction processes in the body in addition to the general reactions exhibited by the respiratory, circulatory, and nervous systems. Increasing the oxygen concentration at rest resulted in a decrease in the volume of pulmonary ventilation and an increase in oxygen uptake, but the volume of carbon dioxide remained unchanged. The pulse rate during exercise declined rapidly as oxygen concentration was

⁵⁰Erling Asmussen and Marius Nielsen, "Pulmonary Ventilation and Effect of Oxygen Breathing in Heavy Exercise," Acta Physiologica Scandinavica, 43: 365-378, 1958, pp. 376-377.

increased. Under the same conditions, blood pressure, especially systolic pressure, increased as much as eleven fold over that pressure recorded while breathing normal air. Although there was a small reduction in minute ventilation during exercise with the increase of oxygen concentration, this diminution of ventilation was not deemed significant. The percentage of oxygen extracted from the gas mixtures was somewhat reduced, but excreted carbon dioxide exhibited little change. The average percentage of oxygen extracted from room air during exercise was 5.2 per cent contrasted to the 3.9 per cent extracted while breathing 96 per cent oxygen. The average oxygen consumption during exercise while breathing room air was reported as 1.4 liters per minute as compared to 1.2 liters per minute breathing oxygen-enriched air. The respiratory coefficient increased from .90 for work in air to 1.19 for 96 per cent oxygen. Biochemical analyses of blood and urine further convinced these investigators that breathing increased concentrations of oxygen upsets the normal oxidation-reduction processes in the body and leads to an accumulation of products of intermediate metabolism. It was conjectured that prolonged breathing of oxygen results in non gaseous acidosis which was explained by the fact that all the hemoglobin was saturated with oxygen and circulates through the body in its acid form rather than its normal reduced form. It was

suggested that due to this factor, oxygen consumption for oxidative needs proceeds at the expense of the normal oxygen saturation of the plasma and tissues.⁵¹

According to Bartlett and Hertz, oxygen inhalation, theoretically speaking, should be beneficial only before exercise of short duration when the ability to accumulate an oxygen debt is the primary factor in determining the winner. It was further emphasized that humans have no physiological mechanism for the storage of oxygen in great quantities and any use of increased concentrations of this gas to alleviate fatigue in prolonged activity or benefit recovery was not supported by scientific evidence. The authors strongly suspect an involvement of psychological factors when evaluating the beneficial effects of pure oxygen on performance.⁵²

Summation of Related Literature

Surveillance of the literature indicated wide disagreement among authors as to the beneficial physiological effects of oxygen inhalation during heavy muscular work. Briggs; Hewlett et al.; Asmussen, Döbeln, and Nielsen; Nielsen and Hansen; A. V. Hill et al.; Barach; Asmussen and Nielsen; Asmussen; and Bannister and Cunningham all report

⁵¹S. A. Brandis, S. A. Iosel'son, and U. N. Pilo-vitskaia, "Functional Changes in the Organism at Rest and Working, During Prolonged Breathing of Gaseous Mixtures With High Oxygen Content," Sechenov Physiological Journal of The U.S.S.R., 46: Nos. 7-12, 935-943, 1960, pp. 937-942.

⁵²Bartlett and Hertz, loc. cit.

that performances were improved as a result of oxygen administration. In contradiction to these results, A. T. Miller et al.; Barrat-Boyles and Wood; and Bartlett and Hertz concluded that oxygen breathing was of no benefit while performing under heavy work load conditions.

Pembrey and Cook; L. Hill and MacKenzie; L. Hill and Flack; Barach; and Brandis et al. reported decreased heart rates during the recovery period following an exercise of severe intensity. A. V. Hill et al. and Åstrand summarized the effect of high oxygen pressure on the heart by suggesting that the action of the heart muscle is regulated, at least partially, by the oxygen tension in the coronary vessels or via the central nervous system which in turn responds to the increased pressure in arterial blood.

A number of studies demonstrated that pulmonary ventilation decreased significantly as oxygen concentration was raised and as the requirements of exercise became more demanding. Bannister and Cunningham attempted to explain this observation as being a result of the depressing effect of oxygen upon the respiratory regulating chemoreceptors. Hickam et al. and Comroe and Schmidt supported the theory that the respiratory center reacts to a disturbance in normal arterial oxygen equilibrium via certain neurogenic stimuli which become more active as the strenuousness of exercise increases. Asmussen and Nielsen proposed that some unidentifiable substance released in the working muscles during

strenuous exertion gave rise to pulmonary ventilation and that increased arterial oxygen pressures reduced the formation of this substance, thus lowering ventilation. Asmussen and Nielsen suggested it was probable that reflexes from muscles and joints of moving limbs contributed to the increase in ventilation and that higher oxygen concentrations depressed these reflexes during exercise. Åstrand reported a summary of the literature pertaining to oxygen inhalation during severe work and indicated that the lowering of pulmonary ventilation was more likely to involve oxygen tension changes in the circulatory system and stimulation of receptors via the blood, rather than nervous impulses from muscles or motor centers. Dripps and Comroe found that resting pulmonary ventilation decreased at first and then increased significantly as a result of prolonged oxygen breathing. It was suggested that this increase in ventilation was due to nervous stimuli, as oxygen sensitive chemoreceptors have only minimal activity under normal conditions and that only during situations of extreme stress is their role in the regulation of respiration fully realized. Baker and Hitchcock believed that the rapid rise in ventilation and respiratory rate was due to the stimulus of a higher carbon dioxide tension and a lower blood pH on the medullary respiratory center.

If the ventilation is decreased and oxygen extraction remains the same or increases, then not only is the individual breathing more economically, but this also implies the possibility for a greater maximal oxygen intake. Unfortunately, data regarding oxygen consumption while breathing oxygen-enriched gas mixtures were infrequently reported.

Although A. V. Hill et al.; Briggs; Baker and Hitchcock; and Brandis et al. noted higher oxygen intakes at rest and during exercise while breathing pure oxygen, their findings, according to A. V. Hill, are subjective to a high degree of error. The increased possibility of errors in respiratory analysis due to high concentrations of oxygen in the inspired air was discussed by A. V. Hill, Long, and Lupton.

Blood lactic acid was reported reduced during heavy exercise and subsequent recovery as a result of oxygen administration by Nielsen and Hansen; Asmussen and Nielsen; Lundin and Ström; Asmussen; and Bannister and Cunningham. A. V. Hill et al. and Hewlett et al. observed no change in maximum blood lactates.

Effects of oxygen inhalation as reported by the more pertinent studies are summarized briefly in Table 1.

Based on published and unpublished research, it is evident that at present, there is insufficient data to indicate that breathing pure oxygen raises the exercise capacity of an individual who is performing a vigorous exercise or

TABLE 1

SUMMARY OF THE MORE PERTINENT STUDIES: EFFECTS OF BREATHING
PURE OXYGEN DURING REST, EXERCISE, AND RECOVERY

	$\dot{V}_E$	$\dot{V}O_2$	$\dot{V}O_2$	$\dot{V}CO_2$	P_{aO_2}	P_{aCO_2}	O_2 Debt	Heart Rate	Blood Lactate	Perfor- mance
Asmussen, 1950	↓W					↑W			↓W	↑W
Asmussen & Nielsen, 1946	↓W				↑W	↓W			↓W	↑W
Asmussen & Nielsen, 1958	↓W				↑W	↑W				
Åstrand, 1956	↓W									
Baker & Hitchcock, 1957	↑R	↑R	↑R				↓R			
Bannister & Cunningham, 1954	↓W								↓W	↑W
Barach, 1934	↓W							↓W		↑W
Brandis et al., 1960	↓W	↓W	↔W				↑W	↓W		
Briggs, 1920		↓W	↑RW							↑W
Dripps & Comroe, 1947	↑R							↓R		
Hewlett et al., 1926	↓W		↑W	↑W					↔W	
Hill, A.V. et al., 1924	↑W	↑W			↑W		↑Rec			↑W
Hill, L. & Flack, 1909								↓Rec		
Hill & MacKenzie, 1958								↓Rec		↑W
Lundin & Ström, 1947	↓W	↓W							↓W, Rec	
Miller, 1952								↔W, Rec		↔W
Nielsen & Hansen, 1937	↓W				↑W				↓W	↑W
Pembrey & Cook, 1908	↔R							↓W, Rec		↑W
Wood, 1949				↔R						

Key:

Increase ↑

Decrease ↓

No Change ↔

R-Rest

W-Work

Rec - Recovery

aids in his recovery. Although in many studies improvement of physical performance was observed, it is still not possible to rule out the effects of psychological influences.

CHAPTER III

METHODS AND PROCEDURES

Six well-conditioned athletes were selected as subjects for this study. Two of them, G. K. and E. S., were members of the 1963-64 Ohio State University track team, E. P. was a varsity soccer player, R. H. was an outstanding freshman swimmer, and D. B. and F. H. were former varsity competitors in track and swimming and at the time of the experiment, were graduate students at the University. The subjects representing the track team were middle-distance or distance runners (440, 880, mile, two-mile) and had been in training for a minimum of four months. R. H., an individual medley specialist, had been in training for five months and at this time was preparing for the American Amateur Athletic Union Indoor Swimming and Diving Championships. The other three subjects were engaged in vigorous conditioning activities designed primarily to maintain strength and cardiovascular endurance. These volunteers ranged in ages from 18 to 28; in height from 68.25 to 78.00 inches; and in weight from 150.0 to 197.5 pounds. The age, height, and weight of each subject appears in Table 2. Subjects were rotated in terms of the two conditions by incorporating the Latin Square Method.

TABLE 2
AGE, HEIGHT, AND WEIGHT OF SUBJECTS

Subject	Age	Height	Weight
D.B.	25	71.00	160.0
B.H.	18	78.00	197.5
F.H.	28	71.75	163.0
G.K.	19	70.00	150.0
E.P.	19	68.25	162.5
E.S.	20	68.75	143.5

Apparatus

Bicycle ergostat. The bicycle ergometer, a Jacquet Universal Ergostat, was the apparatus used for performing the exercise. This stationary bicycle could be calibrated from a low of five watts at thirty revolutions per minute to a high of 450 watts at ninety revolutions per minute. Each subject rode the ergometer for five minutes at sixty revolutions per minute and at a work load of 275 watts* for each of the two experimental conditions. The desired rate of pedal revolutions was maintained by the subject observing an electrically synchronized unit placed directly in front of the bicycle and connected to the sprocket wheel.

*275 watts = .3685 horsepower = 28.0418 kgm/sec.

Gas reservoirs and collecting equipment. Three Collins Chain-Compensated spirometers, with capacities of 120, 350, and 600 liters respectively, with electric kymograph drums and meter sticks attached, contained the volume of inspired gas to be consumed by the subject during rest, exercise, and recovery. Douglas bags with capacities of 50, 100, 150, and 200 liters were connected in series and served to collect the total volume of exhaled air.

Gas analyzers. Gas samples were analyzed for nitrogen percentage on the VanSlyke apparatus and for oxygen percentage on a Beckman Oxygen Analyzer.

Telemetering equipment. Heart rates were recorded by a telemetering system. Patch electrodes were placed on the subject's sternum and on the upper portion of the lateral left rib cage. Electrical lead wires were connected between the electrodes and a transmitter, which was strapped to the subject's back. With the aid of an F.M. tuner, the signal from the transmitter was received and amplified, then recorded on a Sanborn Viso-Cardiette, Model 51, Electrocardiograph.

Procedures

Every subject, in addition to being in excellent physical condition, was completely oriented to laboratory procedure. All of these men had previously served as subjects for one or more studies involving determination of oxygen consumption while performing heavy work loads on the bicycle

ergometer. In addition, each subject was allowed one practice run on the ergometer, during which he familiarized himself with the work load to be used in this study.

Upon entering the laboratory, the subject changed to running clothes which consisted of flat, rubber-soled canvas shoes, shorts, and a T-shirt. He then sat on a training table while the telemetering apparatus was attached and the breast plate adjusted to hold the mouth-piece (Douglas valve).

Pre-oxygenation and rest period. Regardless of working on air or 100 per cent oxygen, the subject spent fifteen minutes breathing from the spirometers before resting expired gas was collected. The purpose of this was twofold: (1) it provided an adequate pre-resting period, and (2) it permitted washing out of the subject's alveoli.

During the five-minute rest period at which time the expired gases were directed into Douglas bags, heart rate was recorded the last ten seconds of the first and fifth minute.

Exercise and recovery. During the exercise period heart rate was recorded the last ten seconds of each minute. During the thirty-minute recovery period heart rate was recorded the last ten seconds of the second minute and the last ten seconds of every fifth minute thereafter.

The gas mixture, whether 100 per cent oxygen or air, was breathed from the 1070 liter reservoir contained in the

three spirometers; the exhaled gas was directed into Douglas bags. In every instance the 1070 liter reservoir did not contain sufficient volume and it was, therefore, necessary to replenish the system. This was done by refilling one spirometer while the subject was connected to another. At all times effort was made to maintain the subject naive to the experimental condition.

Computing gas volumes. Both inspiratory and expiratory gas volumes were measured. Inspiratory volumes were measured by placing a piece of masking tape across the meter stick at either of two instances: (1) when a Douglas bag became filled and expired gas had to be directed into another bag, and (2) when a spirometer was nearly empty requiring the valves be turned so the subject was directed into a fresh reservoir. Exhaled volumes were metered in the 350 liter gasometer.

Gas analysis. The medical oxygen in these experiments contained between .2 and .5 per cent nitrogen. This gas was withdrawn from the spirometer reservoirs during the last minute of the tenth minute of recovery (midway through the experiment) in 50 ml syringes for duplicate analysis. Atmospheric air was assumed to contain: oxygen, 20.93 per cent; carbon dioxide, .03 per cent; and nitrogen, 79.02 per cent. Samples of exhaled air were withdrawn immediately in one liter rubber sample bags and analyzed for oxygen content. All gas volumes were corrected to STPD.

Examples of work sheets utilized in calculating respiratory ventilation volumes and oxygen consumptions for rest, work, and recovery are tabled in Appendix A.

CHAPTER IV

ANALYSIS OF DATA

Description of Data

The gross oxygen cost was computed in milliliters and the results are summarized in Table 3. To determine whether a significant difference occurred between gross oxygen consumptions for the two conditions, the analysis of variance for several matched groups, as described by Edwards,⁵³ was utilized. In order to further demonstrate the effects of oxygen breathing during these experiments, ventilation volumes and total oxygen consumptions for exercise and recovery and resting $\dot{V}_E$ and $\dot{V}O_2$ for each of the two conditions were compared in terms of per cent increase and decrease.

Mean heart rates for each time period appear in Table 4 and are graphically portrayed in Figure 1. To ascertain if there were any significant differences among mean heart rates due to oxygen administration, six different time intervals and their representative mean heart rates for each condition were selected and analyzed using the t test of significance.

⁵³Allen L. Edwards, Statistical Analysis for Students in Psychology and Education (New York: Rinehart and Company, Inc., 1946), pp. 229-231.

TABLE 3
GROSS OXYGEN COST FOR EACH OF
TWO CONDITIONS IN ML.

Subject	Air	O ₂
D.B.	31,110.75	35,064.11
F.H.	32,817.86	34,859.66
B.H.	36,863.38	32,647.26
G.K.	31,293.89	31,986.78
E.P.	33,565.28	44,793.73
E.S.	35,287.62	38,698.39
Total	200,938.78	218,049.93
Mean	33,489.80	36,341.66

Analysis of variance. The results of the analysis of variance for gross oxygen cost appear in Table 5. Appendix G contains the computations for the analysis of variance. An F of 1.189 for gross oxygen cost for rows was obtained which is not statistically significant. This means that the subjects, in relation to one another, did not vary in their performances of each condition. For the columns, an F of 1.922 was obtained which is also not significant. This demonstrates that there was no difference in gross energy expenditure between the two conditions. In other words, the results of the analysis of variance show clearly the homogeneity of variances of the subjects in relation to one another and also between the conditions.

TABLE 4

MEAN HEART RATES (BEATS/MIN.) PER TIME PERIOD AND STANDARD DEVIATIONS

Condition		Time in Minutes													
		Rest		Exercise						Recovery					
		1	5	6	7	8	9	10	12	15	20	25	30	35	40
Air	Mean	79	80	154	167	173	177	177	126	107	100	101	96	98	100
	18.61	13.14	9.91	8.32	8.67	9.13	11.30	16.76	11.92	14.44	14.55	12.43	12.24	11.41
O ₂	Mean	70	79	149	160	168	173	176	118	102	95	92	91	88	90
	8.45	9.87	9.56	12.22	12.35	9.46	9.18	18.12	11.57	13.51	10.58	11.06	14.53	10.13

FIG 1: MEAN HEART RATES PLOTTED FOR 5 MINUTES OF REST, 5 MIN. EXERCISE, AND 30 MIN. OF RECOVERY

TABLE 5
ANALYSIS OF VARIANCE-GROSS OXYGEN COST

Source of Variation	Sum of Squares	Degrees of Freedom	Mean Square	F
Between Columns (Conditions)	24,399,287.85	1	24,399,287.86	1.922
Between Rows (Subjects)	75,502,675.45	5	15,100,535.09	1.189
Residual (Error)	63,483,316.30	5	12,696,663.26	
Total	163,385,279.61	11		

Between Columns.....	F_{01}	- 16.26
.....	F_{05}	- 6.61
Between Rows.....	F_{01}	- 10.97
.....	F_{05}	- 5.05

Gross cost, net cost, and resting $\dot{V}O_2$. In order to provide a more complete analysis of the effects of oxygen inhalation on respiratory adjustment during these experiments, gross cost, net cost, and resting oxygen consumption per minute were determined. These respiratory parameters were compared to the control (air) condition utilizing per cent increases and decreases. Resting $\dot{V}O_2$ and net oxygen consumption are tabled in Appendixes B and C respectively and along with gross oxygen consumption, are graphically portrayed in Figure 2. The bar graphs of Figure 2 reveal a substantial increase in resting $\dot{V}O_2$, an insignificant

Fig 2 Percentage Increases and Decreases in Resting $\dot{V}O_2$, Gross Oxygen Cost, Net Oxygen Cost, Resting Minute Ventilation Volume, and Exercise and Recovery Ventilation Volume

increase in gross $\dot{V}O_2$ and a decrease in net $\dot{V}O_2$ as a result of oxygen inhalation. The explanation for such an exaggeration in resting $\dot{V}O_2$ as a result of breathing pure oxygen cannot be satisfactorily explained.

Previous research⁵⁴ has indicated that resting $\dot{V}O_2$ increases during oxygen breathing, but when the subject is returned to normal air, this parameter declines rapidly to its normal resting level. It was believed that the explanation for this increment in $\dot{V}O_2$ was based on the fact that, as the partial pressure of oxygen is increased, more oxygen is physically dissolved in intracellular and extracellular body fluids. It has also been theorized that an increase in oxygen consumption at rest could be attributed to man's ability to store quantities of oxygen. This ability of oxygen storage has been observed to increase linearly with increases in oxygen pressure, lung volume, and atmospheric pressure. Rahn demonstrated that filling the lungs to capacity after breathing 100 per cent oxygen increased the oxygen storage quantity to 4500 ml (STPD) in a 70 kg. man over that of the normal quantity stored of 370 ml. However, it has been stated that this increased storage ability is based on voluntary apnea after oxygen inhalation and does not occur to such a degree in rest and even to a lesser

⁵⁴Baker and Hitchcock, loc. cit.

degree in work.⁵⁵ Although the lungs, compared to hemoglobin, myoglobin, and the tissues, seem to be the most important area for the storage of oxygen during rest and work, the quantity of oxygen stored diminishes during normal respiration and it is, therefore, with reservation that one considers lung storage of oxygen as a possible explanation of increased resting $\dot{V}O_2$. There is also the possibility that the subjects were not given sufficient duration to establish respiratory equilibrium with the new gaseous environment, caused by increased oxygen pressure. If this is true, then it would be difficult to ascertain the quantity of oxygen dissolved in tissue opposed to that used for metabolic needs. It is, therefore, conjectured that increased $\dot{V}O_2$ obtained during rest in these experiments might be a result of more oxygen being physically dissolved in blood and tissue or occluded in the body in some manner. If the high resting oxygen consumption per minute obtained during oxygen breathing in these studies is a manifestation of metabolic need plus the increased amount dissolved in blood and tissue, then perhaps the pre-oxygenation time of fifteen minutes was of inadequate duration.

⁵⁵Hermann Rahn, "Oxygen Stores of Man," Studies in Pulmonary Physiology, Mechanics, Chemistry, and Circulation of the Lung, Vol. 1 (Biomedical Laboratory, 6570th Aerospace Medical Research Laboratories, Aerospace Medical Division, Air Force Systems Command, Wright-Patterson Air Force Base, Ohio, 1963), p. 198.

Gross oxygen consumption increases 8.5 per cent as a result of oxygen inhalation and is not significantly increased as demonstrated by the analysis of variance.

Net oxygen consumption, as indicated by Figure 2, decreased 21.1 per cent due to breathing oxygen-enriched gas mixtures. However, it must be emphasized that this reduction is due to the effect of the substantial increase in resting $\dot{V}O_2$ while breathing oxygen. It is this distorted resting minute oxygen consumption which lowers the net oxygen consumption to questionable values.

Respiratory data procured during the air experiments are consistent and are similar to data collected previously in studies concerned with respiratory analysis during heavy work under ambient conditions.

Resting minute volume and exercise and recovery ventilation volumes. Figure 2 demonstrates the per cent increase and decrease of resting $\dot{V}_E$, exercise V_E , and recovery V_E above and below the control (air) as a result of oxygen administration. These measurements are tabulated in Appendixes D, E, and F respectively.

As illustrated in Figure 2, resting minute ventilation increased 6.72 per cent during oxygen inhalation. As indicated in Table 1, those studies investigating the effect of oxygen breathing on resting minute ventilation agree with this finding. It was postulated by Dripps and Comroe that the stimulating effect of increased oxygen

pressures on resting minute ventilation could be attributed at least in part, to the increased partial pressure of carbon dioxide at the tissue level. This was believed to occur because of the decrease of available reduced hemoglobin for oxygen transport, an increase in medullary partial pressure of carbon dioxide as a result of cerebral vasoconstriction, an improvement in arterial oxygen tension at the medullary center, or finally, by a lower respiratory tract reflex initiated by the irritating effect of pure oxygen. The effect of oxygen on resting minute ventilation can be summarized in terms of the regulating factors of respiration. Utilizing the most recent data on respiratory control, it seems that the oxygen sensitive chemoreceptors are continually active and stimulate, through the carotid body, the medullary respiratory center as changes in oxygen concentration take place in the blood and at the tissue level.⁵⁶

Exercise and recovery ventilation volumes were shown in Figure 2 to decrease 15.9 and 9.99 per cent respectively. Although the studies which were summarized in Table 1 did not compare per cent increase or decrease in exercise and recovery ventilation volumes as a result of oxygen and air breathing, most of them reported significant decreases in exercise pulmonary ventilation and a minor decrement in ventilation during recovery. A number of possible solutions

⁵⁶Dripps and Comroe, loc. cit.

have been offered to explain this decrease in ventilation volume during exercise and recovery. All have been associated with respiratory control and although the specific regulating factors of respiration during exercise and the preliminary phases of the recovery process are still unknown, it is interesting to note the various theories which are presently employed to explain the effects of these factors. It has been postulated that during exercise under ambient conditions an unidentifiable substance associated with anaerobic work is produced in the working muscles which stimulates the respiratory center directly or indirectly through its action on the chemoreceptors.⁵⁷ Increasing oxygen concentration was believed to diminish the production of anaerobic metabolites, thus lowering ventilation. Another regulating factor to consider is the increase in arterial oxygen concentration as a result of oxygen breathing. It was believed that during exercise there is a sufficient diminution of oxygen tension to activate the chemoreceptors to increase pulmonary ventilation. Administration of oxygen raises oxygen tension to values in excess of the threshold of the carotid and aortic chemoreceptors and the "hypoxic" drive of exercise is never realized.⁵⁸ However,

⁵⁷Asmussen and Nielsen, loc. cit., "Studies on the Regulation . . ."

⁵⁸Åstrand, loc. cit., "Human Physical Fitness . . ."

it has been stated that the reduction of arterial PO_2 during work under atmospheric conditions is too diminutive to stimulate the chemoreceptors and thus, oxygen administration, should have no effect.⁵⁹ Instead it was proposed that nervous stimuli from the working muscles and joints affect the respiratory center and increasing oxygen concentration acts indirectly on this center via these joint and muscle reflexes.⁶⁰ In summarizing recent data, the human chemoreceptor system seems to play the major role in regulating respiration during severe exertion and thus is indirectly responsible for the decrease in ventilation during work and recovery while breathing 100 per cent oxygen. It is believed that the rapid ventilatory response to increased arterial oxygen saturation is too immediate to be explained in terms of changes in the concentration of some substances in the blood, but instead is more likely due to neurogenic factors or chemoreceptor stimulation.⁶¹

Heart rates. To determine whether any significant difference existed between heart rates for each of the two conditions, the t test was utilized. Mean heart rates for both conditions from six different time intervals during rest, exercise, and recovery were selected and analyzed.

⁵⁹Hill, Long, and Lupton, loc. cit., Parts IV-VI.

⁶⁰Åstrand, loc. cit. "Human Physical Fitness . . ."

⁶¹Hickam et al., loc cit.

The similarity of heart rates recorded during exercise and recovery for both conditions suggests that oxygen does not diminish the strenuousness of the performance of the work load. The results of the analysis of heart rates are in accordance with those results obtained after gross oxygen cost for both conditions was statistically analyzed. In other words, these data agree in demonstrating that the energy expenditure of the exercise while breathing air or oxygen is equivalent.

CHAPTER V

SUMMARY AND CONCLUSIONS

Summary

Gross oxygen consumption was determined on six male athletes who exercised on a bicycle ergometer, at sixty revolutions per minute for five minutes at a work load of 275 watts per minute under two different experimental conditions. The first condition involved resting, performing the work load, and then recovering on room air. The second condition was exactly as the first, with the exception that 100 per cent oxygen was substituted in the inspired air. The total exercise time which elapsed for each of the conditions was five minutes.

The open circuit method was utilized in which the subjects breathed, depending on the conditions, either room air or 100 per cent oxygen from three spirometers and exhaled into a series of Douglas bags. Each subject was required to inhale either room air or pure oxygen for fifteen minutes prior to the five minute resting period. The purpose of this fifteen minute period was two-fold in nature: (1) it provided an adequate pre-resting period, and (2) it allowed the subject, during the oxygen experiments to wash out the lungs. Based on previous research

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and recommendations, this pre-oxygenation period was thought to be of sufficient duration to reach an equilibrium between the increased alveolar PO_2 and the oxygen in blood and tissue.

The protocol of the study dictated separate quantification of inspired and expired ventilation volumes and oxygen percentages. Inspired volumes were measured by careful reading of the meter stick attached to each spirometer and nitrogen percentage of these volumes was analyzed on a Van Slyke Apparatus. Expired volumes were metered in the 350 liter spirometer. Samples of expired gas were analyzed for oxygen content on a Beckman Oxygen Analyzer. Total time elapsed for an entire experiment, including the pre-oxygenation period was fifty-five minutes. Percentages of increase or decrease in pulmonary ventilation volumes and oxygen consumption, due to oxygen inhalation are briefly summarized.

Heart rates were recorded throughout the experiment for the purpose of demonstrating the effect of 100 per cent oxygen on the strenuousness of the work for the subjects, as well as a guide to their recovery.

Analysis of variance was employed to ascertain any significant differences in the gross oxygen consumption between the two conditions and between subjects in relation to their individual reactions to these conditions. An F of

1.922 was obtained for conditions and was not statistically significant. For subjects, an F of 1.189 was recorded and does not approach the .05 level of confidence.

In order to compare any changes in heart rate that might result due to oxygen breathing, representative mean heart rates for each of the two conditions were analyzed by the t test. The results of these individual t tests indicate no significant difference between the representative mean heart rates and implies that the strenuousness of the exercise and subsequent recovery period were similar under each of the experimental conditions.

Conclusions

On the basis of the data obtained in this study, the following conclusions were reached:

1. No significant difference was found in gross oxygen consumption between the air and oxygen conditions.
2. In accordance with previously reported data, resting oxygen consumption per minute was substantially increased as a result of breathing 100 per cent oxygen. This may be due to continuing solution of oxygen in blood and tissues after the fifteen minute equilibration period.
3. A decrease was noted in net oxygen consumption while breathing pure oxygen which may be due to the possible error in resting oxygen consumption mentioned in 2.
4. Resting minute ventilation volume increased

slightly and exercise and recovery ventilation declined when oxygen was substituted for air.

5. Although heart rate decreased during oxygen inhalation, no significant difference was noted between representative mean heart rates of the two conditions.

6. Analysis of gross oxygen consumption and mean heart rate suggests that regardless whether 100 per cent oxygen or normal air is breathed, the subject's ability to perform a heavy work load and recover from exertion is unchanged.

Recommendations

As a result of the data obtained in this study, it is recommended that:

1. More intensive research be conducted concerning resting minute oxygen consumption in order to repudiate or sustain the unusual data collected in this study.

2. Further investigations study the possibility of a longer pre-oxygenation period or vary the duration of this period before respiratory observations are conducted in order to establish the optimum wash-out time. It is conjectured that the pre-oxygenation period utilized in this study might have been too short in duration to provide adequate pre-oxygenation, thus affecting the respiratory analyses, especially during the early stages of the experiment.

3. Analysis of exhaled carbon dioxide content be carried out to determine whether it is represented in sufficient quantity to affect the expired oxygen percentages.

APPENDIX A

SAMPLE WORK SHEETS FOR THE CALCULATION OF RESPIRATORY VENTILATION VOLUME AND OXYGEN CONSUMPTION

Condition: Oxygen Subject: D.B. Date: 3/3/64

P_B : 742.4 Spirometer - T_S - Correction Factor - Bell Factor

Inspired: 120 l. - 27.0°C. - .857 - 133.2 cc/mm
 350 l. - 26.2°C. - .861 - 326.1 cc/mm
 600 l. - 26.9°C. - .857 - 517.8 cc/mm
 Expired: 350 l. - 26.0°C. - .862 - 326.1 cc/mm

INSPIRED SIDE

	<u>Meter Stick Distance (mm)</u>	<u>Spirometer (liters)</u>	<u>Uncorrected Volume (ml)</u>	<u>Corrected Volume, STPD (ml)</u>	<u>% O₂</u>	<u>VO₂ (ml)</u>
Rest	280.0	120	37,296.00	31,962.67	99.30	31,738.93
Exercise, Bag 1	654.0	350	213,269.40	183,624.95	99.30	182,339.58
Exercise, Bag 2	351.0	350	114,461.10	98,551.01	99.30	97,861.15
Exercise, Bag 2	92.5	600	47,896.50	41,047.30	99.30	40,759.97
Recovery, Bag 3	537.0	600	278,058.60	238,296.22	99.30	236,628.15
Recovery, Bag 4	359.0	600	185,890.20	159,307.90	99.30	158,192.75

APPENDIX A (continued)

EXPIRED SIDE

Rest	108.0	350	35,218.80	30,358.61	95.12	28,877.11
Exercise, Bag 1	634.0	350	206,747.40	178,216.26	94.30	168,057.93
Exercise, Bag 2	505.0	350	164,680.50	141,954.59	94.46	134,090.31
Recovery, Bag 3	851.0	350	277,511.10	239,214.57	95.05	227,373.45
Recovery, Bag 4	525.0	350	171,202.50	147,576.56	95.43	140,832.31

Differences: VO_2 (ml)

Resting VO_2 (ml), 5 Min.

Rest	2,861.82
Exercise, Bag 1	14,281.65
Exercise, Bag 2	4,530.81
Recovery, Bag 3	9,254.70
Recovery, Bag 4	17,360.44

$$2,861.82 \times 7$$

$$= 20,032.74$$

45,427.60	Gross Oxygen Cost
20,032.74	Total Resting VO_2
25,394.86	Net Oxygen Cost

APPENDIX A (continued)

Condition: Air Subject: D.B. Date: 3/26/64

P_B: 737.4 Spirometer - T_S - Correction Factor - Bell Factor

Inspired:	120 l.	- 26.2°C.	-	.855	-	133.2 cc/mm
	350 l.	- 26.8°C.	-	.852	-	326.1 cc/mm
	600 l.	- 26.4°C.	-	.854	-	517.8 cc/mm
Expired:	350 l.	- 26.8°C.	-	.852	-	326.1 cc/mm

INSPIRED SIDE

	<u>Meter Stick Distance (mm)</u>	<u>Spirometer (liters)</u>	<u>Uncorrected Volume (ml)</u>	<u>Corrected Volume, STPD (ml)</u>	<u>% O₂</u>	<u>VO₂ (ml)</u>
Rest	332.5	120	44,289.00	37,867.10	20.93	7,925.58
Exercise, Bag 1	601.0	350	195,986.10	166,980.16	20.93	34,948.95
Exercise, Bag 2	365.0	350	119,026.50	101,410.58	20.93	40,244.82
Exercise, Bag 2	205.5	600	106,407.90	90,872.35	20.93	
Recovery, Bag 3	491.0	600	254,239.80	217,120.79	20.93	45,443.38
Recovery, Bag 4	285.2	600	147,676.56	126,115.78	20.93	42,688.17
Recovery, Bag 4	683.5	120	91,042.20	77,841.08	20.93	

EXPIRED SIDE

Rest	135.5	350	44,186.55	37,646.94	16.97	6,388.69
Exercise, Bag 1	596.0	350	194,355.60	165,590.97	16.70	27,653.69
Exercise, Bag 2	683.0	350	222,726.30	189,762.81	17.11	32,468.42
Recovery, Bag 3	787.5	350	256,803.75	218,796.80	17.74	38,814.55
Recovery, Bag 4	717.0	350	233,813.70	199,209.27	16.705	33,277.91

APPENDIX A (continued)

	Differences: VO ₂ (ml)	Resting VO ₂ (ml), 5 Min.
Rest	1,536.89	1,536.89 x 7
Exercise, Bag 1	7,295.26	= 10,758.23
Exercise, Bag 2	7,776.40	
Recovery, Bag 3	6,628.83	
Recovery, Bag 4	9,410.26	
	<hr/>	
	31,110.75	Gross Oxygen Cost
	10,758.23	Total Resting VO ₂
	<hr/>	
	20,352.52	Net Oxygen Cost

APPENDIX B

RESTING OXYGEN CONSUMPTION PER MINUTE FOR EACH OF TWO CONDITIONS IN ML.

Subject	Air	O ₂
D.B.	307.38	467.78
F.H.	384.85	598.22
B.H.	474.01	645.28
G.K.	303.15	414.27
E.P.	390.13	535.54
E.S.	396.27	817.43
Total	2,255.79	3,478.52
Mean	375.97	579.75

APPENDIX C

NET OXYGEN COST FOR EACH OF TWO CONDITIONS IN ML.

Subject	Air	O ₂
D.B.	20,352.52	18,691.95
F.H.	19,348.11	13,921.82
B.H.	20,273.17	10,062.53
G.K.	20,683.64	17,487.19
E.P.	19,910.59	26,049.69
E.S.	21,418.03	10,088.34
Total	121,986.06	96,301.52
Mean	20,331.01	16,050.25

APPENDIX D

RESTING (5 Min.) EXPIRED VENTILATION
VOLUME PER MINUTE FOR EACH OF TWO
CONDITIONS IN ML.

Subject	Air	O ₂
D.B.	7,529.39	7,562.74
F.H.	8,435.03	10,650.17
B.H.	10,794.17	10,013.72
G.K.	7,528.02	8,047.82
E.P.	7,511.91	6,445.82
E.S.	8,198.55	10,635.03
Total	49,997.07	53,355.20
Mean	8,332.85	8,892.55

APPENDIX E

EXERCISE (5 Min.) EXPIRED VENTILATION VOLUME FOR EACH OF TWO CONDITIONS IN ML.

Subject	Air	O ₂
D.B.	355,353.78	288,869.62
F.H.	374,198.45	321,618.08
B.H.	310,894.61	266,280.22
G.K.	262,365.39	209,243.37
E.P.	296,522.73	251,442.66
E.S.	373,988.44	321,041.54
Total	1,973,323.40	1,658,495.49
Mean	328,887.23	276,415.92

APPENDIX F

RECOVERY (30 Min.) EXPIRED VENTILATION VOLUME FOR EACH OF TWO CONDITIONS IN ML.

Subject	Air	O ₂
D.B.	418,006.07	363,000.30
F.H.	470,295.55	432,064.24
B.H.	409,841.18	425,512.40
G.K.	317,292.04	290,617.71
E.P.	479,237.22	416,200.13
E.S.	659,252.88	551,372.49
Total	2,753,924.92	2,478,767.27
Mean	458,987.49	413,127.88

APPENDIX G

SUM OF SQUARES COMPUTATIONS FOR GROSS OXYGEN COST IN ML.

Total Sum of Squares

$$\begin{aligned}
 &= EX_2 - \frac{(EX)^2}{N} \quad (\text{correction}) \\
 &= 14,792,680,205.22 - \frac{175,551,539,107.46}{12} \\
 &= 14,792,680,205.22 - 14,629,294,925.62 \\
 &= \underline{\underline{163,385,279.61}}
 \end{aligned}$$

Sum of Squares Between Columns (conditions)

$$\begin{aligned}
 &= \frac{(EX_1)^2}{n} + \frac{(EX_2)^2}{N} - \frac{(EX)^2}{N} \\
 &= 14,653,694,213.48 - 14,629,294,925.62 \\
 &= \underline{\underline{24,399,287.86}}
 \end{aligned}$$

Sum of Squares Between Rows (subjects)

$$\begin{aligned}
 &= \frac{(EX_a)^2 + (EX_b)^2 + (EX_c)^2 + (EX_d)^2 + (EX_e)^2 + (EX_f)^2}{r} \\
 &\quad - \frac{(EX)^2}{N} \\
 &= \frac{29,409,595,202.13}{2} - 14,629,294,925.62 \\
 &= \underline{\underline{75,502,675.45}}
 \end{aligned}$$

APPENDIX G (continued)

Residual Sum of Squares (error)

$$= \text{Total Sum of Squares} - (\text{Between Columns} + \text{Between Rows})$$

$$= 163,385,279.61 - (24,399,287.86 + 75,502,675.45)$$

$$= 163,385,279.61 - 99,901,963.31$$

$$= \underline{\underline{63,483,316.30}}$$

APPENDIX H

t RATIO COMPUTATIONS OF HEART RATES FOR SIX DIFFERENT TIME INTERVALS

$$\underline{t} = \frac{\bar{X}_1 - \bar{X}_2}{\sqrt{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}}$$

Sixth Minute (first minute, exercise)

$$\bar{X} \text{ Air} = 154$$

$$s \text{ Air} = 9.91$$

$$\bar{X} \text{ O}_2 = 149$$

$$s \text{ O}_2 = 9.56$$

$$\underline{t} = \frac{154 - 149}{\sqrt{\frac{98.2}{6} + \frac{71.4}{6}}}$$

$$= \frac{5}{\sqrt{16.4 + 11.9}} = \frac{5}{5.32} = \underline{\underline{.940}}$$

Eighth Minute (third minute, exercise)

$$\bar{X} \text{ Air} = 173$$

$$s \text{ Air} = 8.67$$

$$\bar{X} \text{ O}_2 = 168$$

$$s \text{ O}_2 = 12.35$$

$$\underline{t} = \frac{173 - 168}{\sqrt{\frac{75.2}{6} + \frac{152.6}{6}}}$$

$$= \frac{5}{\sqrt{12.5 + 25.4}} = \frac{5}{6.16} = \underline{\underline{.812}}$$

APPENDIX H (continued)

Tenth Minute (fifth minute, exercise)

$$\bar{X} \text{ Air} = 177 \quad \text{Air} = 11.30$$

$$\bar{X} \text{ O}_2 = 176 \quad \text{O}_2 = 9.18$$

$$\begin{aligned} \underline{t} &= \frac{177 - 176}{\sqrt{\frac{127.8}{6} + \frac{84.2}{6}}} \\ &= \frac{1}{\sqrt{21.3 + 14.0}} = \frac{1}{5.94} = \underline{\underline{.168}} \end{aligned}$$

Fifteenth Minute (fifth minute, recovery)

$$\bar{X} \text{ Air} = 107 \quad \text{Air} = 11.92$$

$$\bar{X} \text{ O}_2 = 102 \quad \text{O}_2 = 11.57$$

$$\begin{aligned} \underline{t} &= \frac{107 - 102}{\sqrt{\frac{142}{6} + \frac{133.8}{6}}} \\ &= \frac{5}{\sqrt{23.7 + 22.3}} = \frac{5}{6.78} = \underline{\underline{.737}} \end{aligned}$$

Twenty-Fifth Minute (fifteenth minute, recovery)

$$\bar{X} \text{ Air} = 101 \quad \text{Air} = 14.55$$

$$\bar{X} \text{ O}_2 = 92 \quad \text{O}_2 = 10.58$$

$$\begin{aligned} \underline{t} &= \frac{101 - 92}{\sqrt{\frac{164.2}{6} + \frac{112}{6}}} \\ &= \frac{9}{\sqrt{27.4 + 18.7}} = \frac{9}{6.79} = \underline{\underline{1.325}} \end{aligned}$$

APPENDIX H (continued)

Fortieth Minute (thirtieth minute, recovery)

$$\bar{X} \text{ Air} = 100 \qquad \Delta \text{ Air} = 11.41$$

$$\bar{X} \text{ O}_2 = 90 \qquad \Delta \text{ O}_2 = 10.13$$

$$\underline{t} = \frac{100 - 90}{\sqrt{\frac{130.2}{6} + \frac{102.6}{6}}}$$

$$= \frac{10}{\sqrt{21.7 + 17.1}} = \frac{10}{6.23} = \underline{\underline{1.605}}$$

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